Acta Pædiatrica 52: 245—251. May 1963

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Intragastric Oxygen and Resuscitation of the Newborn[1]

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Intragastric oxygen was first suggested as a useful technique for oxygenating newborn infants by Ylppö [9, 10]. Following clinical reports by Åkerrén & Fürstenberg in 1950 [1] and later Waller & Morris in 1953 [8], the method was accepted in a number of centers as ideal for newborn resuscitation, primarily because of its safety and simplicity. The following studies were undertaken in an effort to obtain quantitative evidence as to the effectiveness of the procedure in the human infant.

Material and Methods

Nine asphyxiated and 20 normal infants were studied. The clinical descriptions of the asphyxiated group are listed in the accompanying Table. The normal infants were all mature and vigorous at birth.

Two soft polyethylene catheters (Pharmaseal K 32) for the inflow and outflow of oxygen were introduced into the stomach. Gas was administered at one liter per minute, since the recommended flow rate of three to four liters per minute [1] caused the abdomen to become distended and tense. Even one liter per minute proved to be too high a rate for some, and in these the flow was correspondingly reduced. The oxygen content of the gas flowing from the stomach was checked from time to time with an oxygen analyser (Beckman, Model D).

Blood samples for the determination of oxygen saturation [4] were withdrawn from the portal vein, inferior vena cava or left atrium by means of a polyethylene catheter advanced through the umbilical vein. In some, an additional catheter was passed into the aorta, via the umbilical artery and blood pressure recorded with a Statham strain gauge and a direct writing polygraph or a saline manometer.

Because of the importance of maintaining the circulation to ensure maximum distribution of any oxygen absorbed from the gastrointestinal tract, and because of regurgitation of oxygen into the pharynx from where it might diffuse into the lungs, two experimental designs were adopted for the asphyxiated group. In five, (Nos. 1, 2, 3, 4, & 9) a snugly fitting endotracheal tube was inserted and control blood samples were taken during artificial ventilation using the mouth to tube technique with oxygen-enriched pharyngeal air [6]. When the infant was pink and had a good heart beat or blood pressure, intragastric oxygen (I.G. O_2) was com-

[1] This research was supported by the U.S. Public Health Grants H2410 and B2390.

[2] Recipient of Investigatorship of the Health Research Council of the City of New York under Contract I-148.

TABLE 1. *Data on nine asphyxiated infants.*

No.	Apgar score [2]	Age after birth	Sample	Procedure	O_2 sat. %	Arterial blood pressure	Condition of infant	Remarks
1 (ref. 5)	3 at 1 min	98 min	Aorta control	$IPPO_2$	93	25 mmHg mean	Skin pink, tone good	Premature 400 g
				Endotracheal tube blocked and I.G. O_2 started				Death at 2 hours of age
			Aorta	I.G. O_2 2 min	24			
			Aorta	I.G. O_2 5 min	8		Dark cyanosis	Autopsy: No evidence of
				$IPPO_2$ recommenced			Flaccid and motionless	damage to the lung
			Aorta	$IPPO_2$ 3 min	94		Skin pink, moving arms and legs	parenchyma
2	6 at 1 min 1 at 10 min	12 min	P.V. control		7	17 mmHg mean	Occasional gasps, limp, dark cyanosis	Premature 490 g
			P.V.	I.G. O_2 30 min	7		No change	Death at 80 min of age
				Endotracheal tube inserted $IPPO_2$				No autopsy
			P.V. control		80	80 mmHg mean	Skin pink, moving arms and legs	
				Endotracheal tube blocked and I.G. O_2 started				
			P.V.	I.G. O_2 10 min	10		Convulsion	
				$IPPO_2$ recommenced				
			P.V.	$IPPO_2$ 2 min	87		Skin pink, moving arms and legs	
3	3 at 1 min	46 min	Aorta control	$IPPO_2$	66		Limp, cyanotic	Premature 265 g
				Endotracheal tube blocked and I.G. O_2 started				
			Aorta	I.G. O_2 11 min	3		Unchanged	No autopsy
				$IPPO_2$ recommenced				
			Aorta	$IPPO_2$ 3½ min	46		Unchanged	
4	2	53 min	P.V. control	$IPPO_2$	78		Skin pink, tone poor Lungs non-compliant	Weight: 3130 g
				Endotracheal tube blocked and I.G. O_2 started			Clinical diagnosis: Diaphragmatic hernia and hypoplastic lungs	Death at 93 min of age

			P.V.	I.G. O_2 4 min $IPPO_2$ recommenced	2		Dark cyanosis	Autopsy: Diaphragmatic hernia hypoplastic lungs
			P.V.	$IPPO_2$ 5 min	76		Skin pink	
5	5	31 min	Aorta control	$IPPO_2$	59	65/40 mmHg	Cyanotic	Weight: 3840 g Death at 2 hours of age
			Aorta control	Spontaneous breathing 70% O_2	10	35/20 mmHg	Labored respiration, marked chest retraction	Autopsy: Intrauterine pneumonia due to monilia; lesions also in placenta and membranes
			P.V.	I.G. O_2 started I.G. O_2 7 min	10		Steady deterioration	
6	3	20 min	P.V.	Spontaneous breathing 70% O_2	4	25/15 mmHg	Cyanotic, labored respiration, marked chest retraction	Weight: 3900 g
			P.V.	I.G. O_2 started I.G. O_2 12 min	4		No change Steady deterioration	Death at 1 hour of age Autopsy: Massive aspiration of meconium
7	—	30 hours	P.V. control	Spontaneous breathing 70% O_2	46	50/30 mmHg	Cyanotic, tone good, quiet respiration	Weight: 2900 g
			P.V.	I.G. O_2 started I.G. O_2 15 min Spontaneous breathing continuing	34		No change	Death at 24 hours of age No autopsy Clinical diagnosis: tricuspid atresia (angiocardiogram)
8	4	2 hours	Aorta control	Spontaneous breathing 70% O_2	27	32/10 mmHg	Limp and cyanotic	Weight: 2510 g
				I.G. O_2 started I.G. O_2 10 min	28			Death at 3½ hours of age
			Aorta	Spontaneous breathing continuing				Anencephaly
			Aorta	I.G. O_2 1 hour	9	14 mmHg	Steady deterioration	
9	3	10 hours	Aorta control	$IPPO_2$	98	48/30 mmHg	Skin pink, tone poor	Weight: 2460 g
				Endotracheal tube blocked and I.G. O_2 started I.G. O_2 4½ min		90/55 mmHg		Death at 15 hours of age Anencephaly
			Aorta	I.G. O_2 7 min $IPPO_2$ started	5	50/35 mmHg	Convulsions	
			Aorta	$IPPO_2$ 4 min	94			

$IPPO_2$ = ventilation of lungs with intermittent positive pressure oxygen through an endotracheal tube.
I.G. O_2 = intragastric oxygen. P.V. = portal vein.

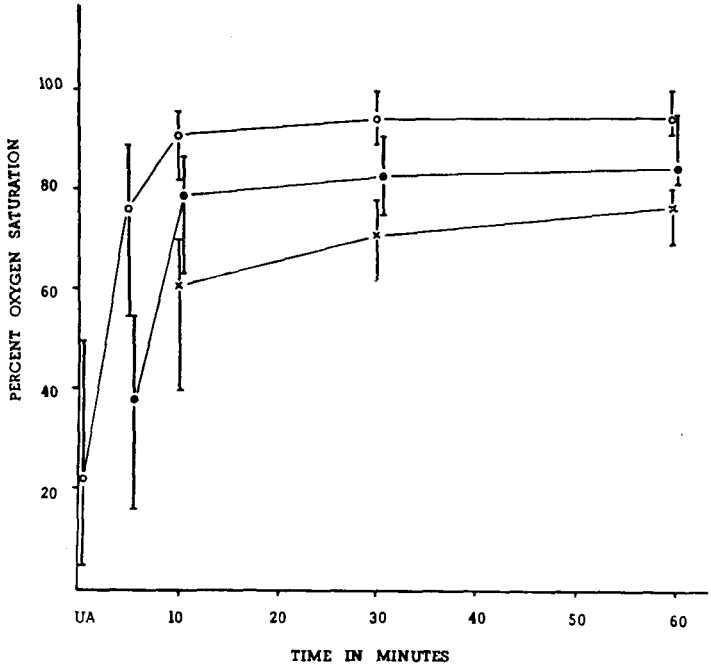

Fig. 1. Percent oxygen saturation in serial blood samples from ten vigorous infants during the first 60 minutes of life. UA = umbilical artery at birth, ○ = left atrium, ● = portal vein, × = inferior vena cava. Range is indicated by vertical lines.

menced. Once gas was flowing back satisfactorily through the outflow tube and the abdomen was distended, artificial ventilation was discontinued and the endotracheal tube occluded. In the remaining four of this group (Nos. 5, 6, 7, & 8), control blood samples were taken with the infant spontaneously breathing 70% oxygen. This environmental oxygen was maintained and the infant continued to breathe while intragastric oxygen was administered.

In ten of the normal control group, serial blood samples were taken from the portal vein, inferior vena cava and left atrium during the first 30 minutes of life, the infant breathing room air. The catheter position was ascertained from the pressure tracing and the blood oxygen saturation. Half of the remaining ten were given I.G. O_2 from within three minutes of delivery. The other half were used as controls and given intragastric nitrous oxide.

Results

Asphyxiated group (see Table 1)

The five infants who were well oxygenated when the lungs were ventilated with oxygen became severely hypoxic when the endotracheal tube was occluded and I.G. O_2 substituted for pulmonary ventilation. They could be readily re-oxygenated when artificial ventilation was reinstituted (Nos. 1, 2, 3, 4, & 9). In the remaining four who were breathing spontaneously (Nos. 5, 6, 7, 8) there was no rise in oxygen level when gastro-intestinal oxygen was given. One infant (No. 7) even showed a fall (46 %→ 34 %) possibly due to splinting of the diaphragm by the distended stomach.

Normal group

The percent oxygen saturation at birth, and during the first 60 minutes of life in

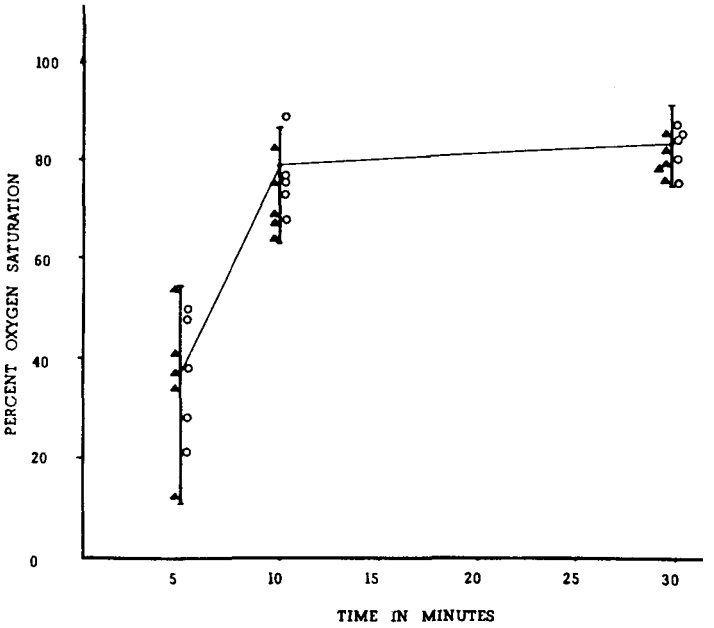

Fig. 2. Percent oxygen saturation in serial portal vein samples in 20 vigorous infants. Continuous and vertical lines = mean values and range for 10 infants breathing room air. O = values for 5 infants receiving I.G. O_2. ▲ = values for 5 infants receiving intragastric nitrous oxide.

the umbilical artery, portal vein, inferior vena cava and left atrium are shown in Fig. 1. It may be seen that the mean value in the portal vein is considerably higher than that in the inferior vena cava. In some instances it was almost equal to that found in the left atrium. When the portal vein oxygen level in these infants is compared with the values found in normal infants receiving intragastric oxygen or intragastric nitrous oxide (Fig. 2) it may be seen that there is no difference. The infants receiving intragastric nitrous oxide remained awake and vigorous and were clinically indistinguishable from those receiving I.G. O_2.

Comment

These studies have shown that when the absorption of oxygen from the lungs is pathologically impaired, or prevented by occlusion of an endotracheal tube, absorption from the gastro-intestinal tract is negligible. Failure of absorption cannot be attributed to circulatory collapse since blood pressure measurements were satisfactory and high oxygen levels were promptly achieved when pulmonary ventilation was recommenced. The gastro-intestinal route therefore cannot be used as an alternative to the lungs for oxygenation of the newborn.

It was of considerable interest to note that even in the previable infants adequate blood levels of oxygen could be maintained by artificial ventilation, and further, that this could be achieved without apparently damaging the lung parenchyma.

Relatively high levels of oxygen in the

portal vein of normal infants did at first sight raise the possibility of some added absorption of oxygen. However, similar values seen in those infants receiving either intragastric oxygen or nitrous oxide indicated that this was unlikely. These high values could be explained by a lower oxygen consumption in the gastro-intestinal tissues or differences in regional circulation times.

Why then has this technique appeared to be of value from the clinical point of view? Regurgitation of 100 % oxygen into the pharynx from where it could diffuse into the lungs, is one possibility. This might account for infants improving in color in the absence of visible respiratory movements. The double lumen catheter usually employed might also be acting as a pharyngeal airway, holding the tongue forward in the limp depressed infant. If this were so, the beneficial effects attributed to I.G. O_2 could be achieved as well by maintaining a clear airway and administering oxygen into the pharynx. Finally, it is important to bear in mind that the chemo-receptors are rugged and continue to stimulate the respiratory center which responds under conditions of severe asphyxia. We have observed well-coordinated deep gasping in an infant with no measurable oxygen in the arterial blood and a pH of 6.5. This ability of the newborn to respond under conditions of severe asphyxia makes it difficult to evaluate a particular resuscitative procedure in the absence of controlled observations.

The use of intragastric oxygen is also not without danger [3]. It may reduce ventilatory movements by pressure on the diaphragm; and rupture of the stomach has been reported [7]. Perhaps the greatest danger is that it lulls the nurse or physician into a false sense of security and prevents him from thinking of, or applying effective ventilation.

Conclusion

Negligible amounts of oxygen are absorbed from the gastrointestinal tract in the human infant. High levels of oxygen observed in the portal vein of normal vigorous infants, are probably related to differences in regional oxygen consumption or circulation.

The tecnique of administering gas into the stomach is not a benign procedure and carries definite dangers. It cannot be considered of any value for resuscitation, nor as an additional source of oxygen for the sick infant. The only effective way to resuscitate a newborn infant is by pulmonary ventilation either through the infant's own efforts, or by the application of intermittent positive pressure.

Summary

The absorption of oxygen from the gastro-intestinal tract was studied in nine asphyxiated and 20 normal infants. There was no evidence of transfer of detectable amounts of oxygen from the gastro-intestinal tract to the systemic blood in the asphyxiated group when absorption from the lungs was pathologically impaired or prevented by occlusion of an endotracheal tube. High oxygen levels were promptly achieved when pulmonary ventilation was instituted. Relatively high levels of oxygen in the portal venous blood of normal infants breathing room air could not be attributed to added absorption from the gut since similar values were seen when intragastric nitrous oxide was given.

The technique of administering gas into the stomach is not a benign procedure and carries definite dangers. It cannot be considered of any value for resuscitation, nor as an additional source of oxygen for the sick infant.

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Submitted Sept. 29, 1962
Accepted Oct. 20, 1962.

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